orraphy, perineorrhaphy, appendectomy, and posterior plication of round ligaments. This patient developed pain in the right lower chest posteriorly on the second postoperative day. This gradually increased in intensity and was aggravated by deep inspiration; no friction rub, bronchial breathing or cough at this time. On the fifth postoperative day the patient developed pain, with marked swelling of right wrist and with pain in the shoulder. On the sixth day there was a severe swelling of the vulva, and pain over course of the right saphenous vein. Slight hacking cough developed on the night of the seventh postoperative day. Color was fair. Became irrational on the eighth postoperative day. Condition continued to become more serious until the tenth day, when death occurred. Death was stated to be due to pulmonic condition. Consent for autopsy was not obtained. This patient was in a very toxic condition the last few days of her life, and from the extent and various locations of complications noted, it seems fair to at least mention the possibility of multiple emboli. Here again the question arises: Was the anesthetic responsible for the death?

CONCLUSIONS

In the foregoing, the results of ethylene-oxygen anesthesia as shown in a short series of two hundred cases have been given While this is a small number, it seems to measure up as an average group.

The complications and disasters following the administration of this anesthetic in this series have been here presented.

The writer's personal impression is that the good results far exceed the bad, and that ethylene has been one of the great advances in medicine of the last few years.

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ANGINA PECTORIS AND ALLIED CONDITIONS*

REPORT OF CASES

By PHILIP KING BROWN, M. D.
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DISCUSSION *by A. L. Bloomfield, M. D., San Francisco; Franklin R. Nuzum, M. D., Santa Barbara; Bernard Kaufman, M. D., San Francisco.*

THE significance of heart pain and its causes, and a definition of what may be included in the syndrome of angina pectoris, merit discussion.

Heberden's description is clear: "They who are afflicted with it are seized while they are walking (more especially if it be uphill and soon after eating) with a painful and most disagreeable sensation in the breast which seems as if it would extinguish life if it were to increase or continue, but the moment they stand still all this uneasiness vanishes. In all other respects the patients are at the beginning of the disorder perfectly well, and in particular have no shortness of breath, from which it is totally different. The pain is sometimes situated in the upper part, sometimes in the middle, sometimes at the bottom of the os sterni. It likewise very frequently extends from the breast to the middle of the arm. The pulse is, at least sometimes, not disturbed by the pain." Heberden goes on to the variations in symptomatology and seems to include left coronary thrombosis, in referring to pain that may last several days.

ALLBUTT AND MACKENZIE CLASSIFICATIONS

Clifford Allbutt described three types of cases:

1. Typical postural pain, radiating to various parts of the body with arrest or restraint of respiration and sense of impending death.

2. Cases with marked atheroma fibrillation and general bodily pain.

3. Aberrant or larval types in which he includes the cases with the fear of death complex, unusual sensory manifestations, pain beginning in heart apex, or epigastric area, or even lower in the abdomen.

Sir James Mackenzie in one of his last published articles before his death (*London Lancet,* 1924, Vol. II, pp. 694-7) referred to two types of angina, the typical Heberden attacks and a mild form commonly found in women. In an analysis of two thousand patients who suffered from heart pain, one thousand cases were eliminated because of unsatisfactory recording of facts. In the other one thousand cases where subsequent events were recorded with precision, he made the following observations.

The cases were first divided: one, into a small group in which the pains were not associated with any disease of the heart; and two, the larger group where it was. The pain and its location, radiation, and other characteristics is the same in both groups.

In Group One he classified heart pain as a reflex manifestation of ill health, similar to vomiting, and which when it affected the nervous system, resulted in angina. The vast majority of this group were women under the age of fifty. During the war the condition was found in young soldiers suffering from obscure infections. Mackenzie followed several hundred cases of "secondary angina" and found that not one had died from any heart affection. The heart symptoms in every case disappeared, many were restored to health, others were crippled from the persistence of the original complaint or from some of its effects.

Some of Mackenzie's followers have taken his viewpoint about mild attacks of heart pain on exertion, and would have us believe that even when it radiates typically a neurosis underlies the conditions found in these patients.

A few cases are presented for the purpose of illustrating these conditions and to form the basis of this discussion.

REPORT OF CASES

CASE 1.—Dr. A., age seventy-one, had his first attack during his strenuous efforts at the time of the San Francisco fire in 1906, the severe substernal pain radiating to the left shoulder and down the left arm. Since then he has had eight major attacks; and if the

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minor attacks which follow smoking, slight but undue effort and indigestion were estimated at only one a day, he would have had over eight thousand such minor experiences. He goes now several days without such attacks, but formerly has had as many as eight or ten in twenty-four hours. Six years ago he had a third type of anginal pain; a left coronary thrombosis, for the pain of which he received a quarter of a grain of morphin every ten minutes for five doses before he got relief. In addition to these three types of anginoid pain he developed an intermittent claudication of the right leg, a further inhibitor of physical effort; and he had suffered from eight attacks of inflammatory edema of the lungs, seven of them at night, which were usually marked by a chill, fever to 104 degrees, pinkish sputum, and râles over both lung bases and fairly well up the back. Digitalis and caffein were indicated.

Such attacks are interpreted as painless spasms of some part of the cardiac vascular system. Wassermann has described right coronary thrombosis as painless, but the suddenness of the edema in this patient marked his condition as an acute failure of the left heart. The most striking point in this patient's history was that this man with three types of anginal pain in the heart area repeatedly said that the pain of his daily attacks, the eight major attacks, and the attack of coronary thrombosis, differed only in intensity and duration, and not in the least as to manner of onset or distribution.

Case 2.—Captain S., age sixty-eight, had precordial pain for ten years, usually induced by effort. There never was fear of impending death in an attack. Nitroglycerin promptly relieved him, and in the minor attacks, standing still resulted in an immediate lessening of the pain, and abatement within a minute or two. His condition was not serious enough to him to cause him to consult a physician until on one occasion, when he had a severe substernal pain, sudden in onset and lasting half an hour. Following a treatment with diuretin (theobromin sodium salicylate), reduction in his diet and weight, and moderation in exercise, there was no recurrence of an attack for six years. In fact he had gone so long without any attack of any kind that he neglected diet and rules of effort. After a hundred-mile automobile ride in the hot sun, he awoke in the night with a severe substernal pain lasting eight hours and of such intensity that when finally a doctor came, one grain of morphin was required to relieve him. Three days later he was still "sore" in the heart area and left wrist. He was confined to bed eight days. While free from pain and attacks now, five weeks later, he moves slowly and with due fear of further trouble.

The pain from his one major attack six years ago, and this last attack which was diagnosed as coronary thrombosis, were the same in substernal origin and left arm radiation, and differed only in duration and intensity. His minor attacks were marked by the onset of pain near the heart apex, spreading over the heart area, and some pain radiating to the left arm and forearm, but not to the left shoulder.

Case 3.—Mr. G. L., age fifty-eight, gave a history of typical substernal pain attacks radiating to both arms; a low blood pressure, especially during attacks; a deflection downward of his T wave in lead one of his electrocardiograph; and was diagnosed as having angina pectoris with marked coronary involvement. He died suddenly in what seemed a sinking attack, apparently painless. Autopsy showed acute extensive thrombosis of his right coronary artery, high up.

Case 4.—Mrs. W., age forty (reported as Case 5 in the last series published by Dr. Walter B. Coffey and the writer), had attacks of numbness in left arm and fingers after exertion, of five to ten minutes' duration. These continued over a period of eleven years before she had a typical attack of angina pectoris, which was marked by substernal pain extending along the base of the heart to the apex and around the left side to a point beneath the angle of the left scapula and then down the inner side of the left arm and forearm and up the left side of the neck. For eighteen hours this pain continued until it was finally relieved by morphin, after a trial without relief, of nitroglycerin. She remained in bed three weeks. After that she had as many as four attacks a week of similar pain, always induced by effort. These attacks were of shorter duration, and were relieved by nitroglycerin. A left cervical sympathectomy two years ago relieved her entirely, and eight days after the operation she walked a mile up a moderate grade, without an attack.

One could go on endlessly enumerating variations in manner of onset, severity of attacks, duration and radiation of pain and pathological conditions detected before and after attacks. It is not strange that great confusion results from the marked differences which characterize attacks rightly called angina pectoris. It is the purpose of this paper to try to clarify the situation somewhat, and to simplify it by calling all these attacks angina pectoris, however mild some of them may be.

The influence of the late Sir James Mackenzie in England and a few optimistic cardiologists in this country was exerted in an effort to minimize the significance of heart pain in certain conditions in which they tried to establish a basis for the diagnosis of mock angina or pseudo-angina. It would seem to be timely to protest against all this, and to state that treatment of substernal or apical heart pain, in my experience, has resulted in postponing the more classical attacks of angina pectoris for periods often ranging from five to ten years. It is very poor satisfaction to say "I told you so" to people who finally have the severe attacks, but who have shopped around for advice to their liking, when they have had minor heart pain with radiation, induced by effort, more apt to occur after eating and relieved by quiet. Mackenzie points to the large group of these patients who die of other conditions, as if that settled the problem. The first patient whose history was outlined in this paper, with his eight thousand minor attacks can hardly be called a false angina when one considers his major attacks and his coronary attack. Nor can the author's longest duration patient, L. W., age seventy-three, who had a major attack at age thirty-five, while driving a trotting horse that had to be held down during a race because of his tendency to break. It was ten years before his second attack and then followed ten years of minor attacks with a few major ones and finally when he came under my care he was almost bedridden. A routine of diet, diuretin, cleaning up infections of teeth and prostate resulted in a relief that led him to spend a winter abroad, and on his seventy-second birthday he wrote a postcard from Yellowstone Park, where he was enjoying himself, free from attacks; but never without his bottle of nitroglycerin, taking it at the first warning of pain, and never waiting to see how severe an attack might be. Perhaps he will die of some other condition. Certainly it would not be proper, on that account, to diagnose

his condition as a false angina. His aorta is big and rigid, and his attacks have the characteristic location and radiation described by Heberden 150 years ago, but they vary greatly in intensity and distribution—the severer ones sometimes even causing right-arm radiation of pain.

CASE 5.—A doctor's wife, Mrs. A., age forty-five, came to me twenty years ago with what had been diagnosed as mock angina. She had a great deal of arthritis and a chronic cholecystitis with achylia. Since then I have succeeded, after much effort and patience, in inducing her to have all her teeth extracted and her gall bladder removed. It took ten years and more to accomplish this. The arthritis, the pain and digestive disturbances were better in consequence. Treatment has relieved greatly the "mock" angina years earlier, but in recent years indulgence in certain foods, especially sweets, has made the gastric symptoms worse and the angina has become more frequent and severe. Medical men in her family now recognize it as a true angina, typical in its relation to effort, with her margin of safety encroached upon by a full stomach. She is relieved of her pain by nitroglycerin, and likely to die in an attack because of her very large aorta and high pressure; in which the sudden exit from coronary thrombosis is less likely to come than though the pressure were very varying or low.

CASE 6.—One more case marks the skepticism or optimism of some medical men and the danger of the gospel of pseudo-angina. A hurried call six years ago took me to the bedside of a surgeon, where he was found attending to some last matters because he had had several attacks of angina pectoris in the few days previous. The usual regimen was instituted, including his parting in due time with some very bad teeth and tonsils which he would not have allowed twenty-four hours in any patient's mouth. The result has been that five years have passed without any more attacks. In the meantime a leg of venison came to me from a 120-pound buck, which was "dragged 300 yards through the brush" without his having any pain. The incident of the old angina was laughed off. One does not want to rub it in, especially to a doctor, so I said nothing except a word of caution about not fooling with his heart at his age. The next attack came, this time with grave symptoms, because in the meantime had occurred the uncovering of a polycythemia. The attack took on more the nature of a coronary thrombosis and was precipitated at the time by taking a little too much phenylhydrazin or taking it too rapidly. There were some transient cerebral symptoms at the time. The fact remains, however, that the warning was enough and there is no more nonsense about false angina.

COMMENT

There is great danger in these conditions of wanting to interpret symptoms too lightly. In medicine it should be the ironclad rule to reverse the stand of law which considers a man innocent till proved guilty and hold symptoms referable to important organs as serious until the fullest investigation shows them to be susceptible of a better interpretation than the serious one first suggested. We have passed through the period of taking some convulsions too lightly, but who would leave a man at his job as a locomotive engineer once he had had a single manifestation of epilepsy? Before Economo made clear the existence of a condition we now know as lethargic encephalitis with its dreadful aftermath in so many cases, and its countless forms of minor ailments, there were many sufferers from that disease who were dismissed as neurasthenics because of their depressed condition, or of the Parkinsonian syndrome or because the many symptoms marked what might easily have seemed a too great dwelling on minor things, and a physical inanition common in asthenic nervous disorders. The recent controversy over the significance of annular shadows in x-ray plates of the lung, interpreted as pleural manifestations, as they were by the early propounders from the leading center in America for the study of lung diseases, is an excellent example of the placing of too little importance on the serious condition of lung cavity, which today everyone believes these shadows to represent.

For these reasons it seems wise that we should regard heart pain more seriously, and potentially consider as cases of Heberden's angina, all cases of heart pain produced by effort and aggravated by smoking, exposure to cold, by diabetes, distended stomach and nervous excitement, or accompanied or not by dyspnea which is largely dependent on the underlying heart condition, as is change in blood pressure during attacks, and which cases are finally relieved by nitroglycerin. If the pain does not radiate, it may still be angina pectoris, and attacks may come in time with more characteristic symptoms.

If the pain be unrelieved by one or two doses of nitroglycerin and even grows more severe and is associated with alarming symptoms, coronary thrombosis must be thought of. Here morphin must be used at once and until relief comes, for the nitrites do no good and may even do harm. The reaction of slight fever, congestive failure, lowered blood pressure, pericardial rub and definite change in the T wave of the electrocardiograph, will confirm the diagnosis.

If the pain instead of being substernal begins in the region of the heart apex and is never substernal, do not be too sure that you have not to do with angina pectoris. The coronary plexus has been shown by Wiggers to have vasoconstrictor fibers, and Humber in the laboratory at the Southern Pacific Hospital has shown that the coronaries in cats contract when the superior cardiac nerve from the superior ganglion and the cardiac nerve of the vagus are stimulated, even ten minutes after the heart ceases beating.

It is by no means established what angina pectoris actually is, but from our studies of cases at the bedside and during the operation of cervical sympathectomy and at autopsy where the coronaries have been injected with lipiodol or barium sulphate and then x-rayed (so as to show changes in coronary circulation, if present, before sectioning the vessels), and from studies on normal animals through recording by every means, it was possible to find such changes as are recordable in heart action under stimulation of the various nerves with a weak electric current, nicotin, and the injections of distilled water; and we have satisfied ourselves that heart pain whether begin-

ning substernally or at the apex is to be regarded seriously and should be studied thoroughly before being passed off as of no consequence.

SUMMARY

For working purposes we propose the following, most of which well-known writers advanced long ago:

1. Typical Heberden's angina begins with substernal pain which radiates to the left arm.

2. Clifford Allbutt refers to larval or aberrant forms where the pain begins in the apex region. Some of our patients who have died in attacks had the pain begin there. There may be coronary disease in such cases which it may not be possible to detect.

3. Abundant evidence has presented itself to us that major and minor attacks and coronary thrombosis may occur in the same patient, with no difference in the point of origin of pain or radiation. Duration and intensity alone vary.

4. If blocking a left coronary artery produces pain in the same locality, radiating to the same parts as do the typical Heberden attacks, then it seems fair to suppose that the coronaries play some part in many attacks of true angina.

5. Some cases of true Heberden attacks have no radiation of pain from the original substernal region. Such patients have sometimes developed them later, or have even died in attacks without true radiations.

6. Right coronary artery thrombosis may be always painless as claimed by Wassermann, and we have had patients where pathological findings seemed to prove this.

7. Typical attacks of Heberden's angina relieved by nitrites may follow coronary thrombosis.

8. Finally it seems to us that the most reasonable explanation of the phenomena of angina pectoris is that of spasm of the first part of the aorta (C. Allbutt) or the coronaries or both. Death occurs when the coronary circulation is too long interfered with by an extensive atheroma which contributes to ischemia.

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DISCUSSION

A. L. Bloomfield, M. D. (Stanford University School of Medicine, San Francisco).—Doctor Brown's paper brings out very clearly the main practical difficulty which the physician encounters in connection with angina pectoris, namely, whether the pain is of serious significance or of no consequence. Confusion seems to have arisen particularly in connection with pain which does not have the typical localization or radiation. This, as Doctor Brown clearly points out, by no means excludes a serious disorder which may terminate fatally. The features which should be relied on in making the diagnosis of serious cardiac pain are more the circumstances under which it arises than the character of the pain itself. If it is clearly related to exercise, excitement or eating, the burden of proof is on the man who says it is not of grave significance. Vague or even severe pains, on the other hand, which occur under other circumstances; for example, when the person is quietly in bed and particularly if there are no other evidences of cardiac disease, require much more careful consideration before one can be sure that they are of real import. While the error of convicting a patient unjustly of angina pectoris is a serious one, the reverse is almost as unfortunate. Above all, the decision should not be made too hastily in a doubtful case, but there should be careful observation of the patient over a period of time and a detailed study of the reaction of the cardiovascular system.

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Franklin R. Nuzum, M. D. (Cottage Hospital, Santa Barbara).—The author's paper emphasizes the diversity of symptoms classified under the symptom complex known as angina pectoris. His illustrative cases demonstrate the contention that angina pectoris is not the name of a disease, but the name given to symptoms varying in location and intensity, and suggesting the varying pathology that may cause these symptoms. He strongly emphasizes the point that even minor attacks of pain may be the result of definite pathologic changes occurring in the root of the aorta, in the coronary vessels, or in the myocardium itself. That minor attacks, or even major seizures, may be due to spasm of the coronary vessels and of spasm of vessels in other parts of the body, is suggested by a marked increase in blood pressure that frequently occurs during an attack of angina pectoris, the pressure falling immediately on the cessation of the attack. This rise in pressure may go to a height of 80 to 100 millimeters of mercury above the patient's usual pressure.

Such an event tends to disprove the time-honored theory that angina pectoris is due to a weakened myocardium. A weakened myocardium would not be so likely to respond with a markedly increased pressure.

As Doctor Brown has stated, those instances in which the pressure is lower than the patient's normal during the attack, which persist for long periods of time, and which require large amounts of morphin sulphate for relief, are cases in which coronary occlusion occurs. Too frequently these attacks are not recognized as being due to coronary occlusion, and such a patient is allowed to get about too quickly. This early activity increases the possibility of rupture of the infarcted area of the myocardium. That rupture of the heart wall occurs much more frequently under such circumstances than has been generally recognized is evident from the increasing number of instances of this kind described in pathological reports.

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Bernard Kaufman, M. D. (2000 Van Ness Avenue, San Francisco).—Doctor Brown's remarks are of distinct service in that he again directs attention to the symptom of precordial pain. The fact that one might not agree in toto with his viewpoint is of minor significance. It is more important that he once more emphasizes the lack of agreement concerning a single important symptom, thus maintaining our interest in its continued investigation.

In this regard it might be well to recall that certain apparently contradictory clinical facts need elucidation before one may safely accept any of the generalizations so far offered in explanation of this symptom.

The following clinical observations might serve to illustrate some of the difficulties with which one is confronted when attempting to generalize concerning precordial pain. From them it is evident that, even when associated with the radiation said to be characteristic of true angina pectoris, this symptom may occur in clinical conditions of a diametrically opposite character.

The precordial pain of the so-called true angina pectoris is said to occur with and to be characterized by a rise in blood pressure. But a similar pain with a similar radiation may arise in conditions associated with a fall in blood pressure. For example, the subjective precordial symptoms seen occasionally during an attack of paroxysmal tachycardia are essentially identical with those associated with true angina pectoris.

It is true that this similarity only becomes marked after the paroxysm of tachycardia has persisted over a certain period and when the attack is of a certain

intensity. When both these conditions are fulfilled the subjective symptoms in both conditions are remarkably similar and are present not only during the attack itself, but also after the attack has cleared up. The significance to be attached to this clinical observation lies in the following: It is often urged that a patient prone to attacks of angina pectoris will cease to have such attacks upon the onset of cardiovascular decompensation, but may again be the victim of their recurrence when the functional activities of the cardiovascular system have once more been restored to normal. The appearance of such precordial pain with its characteristic radiation during a prolonged attack of paroxysmal tachycardia shows that the contrary condition is, at times, also true, namely, that a person may be persistently free from precordial pain until cardiovascular decompensation (that is, with paroxysmal tachycardia) sets in, and then experience the precordial pain only when the cardiovascular decompensation has reached a certain intensity. Further, that such a person becomes and remains free from the precordial pain and its radiation so long as cardiovascular compensation is maintained.

Nor is an attack of paroxysmal tachycardia singular in this regard. A person without the necessary preliminary training, if he persists in severe physical effort, will develop not only a precordial pain with a sense of constriction across the chest, but also coincidentally an acute cardiac dilation with resultant cardiovascular insufficiency and fall in blood pressure.

A similar clinical paradox is seen, when one recalls the oft-repeated statement that patients suffering from angina pectoris cease to have attacks of precordial pain during the febrile state. It cannot be the febrile state alone that is operative in such cases because patients with acute pericarditis and fever not infrequently complain of an intense precordial pain, which pain with its direction of radiation is indistinguishable from that of angina pectoris.

It would therefore seem a fair assumption that the symptom of precordial pain with its customary direction of radiation is not to be looked upon as the sole criterion by which one may safely presume to evaluate either the nature of the condition calling forth the pain, the guide upon which one may safely rely with respect of therapy or the basis upon which one should rely in regard to prognosis.

A similar uncertainty exists in regard to the pain experienced at the apical region of the heart. Doctor Brown has referred to Mackenzie's clinical observations and his resultant opinion regarding the lack of significance to be attached to pain arising in this region. It is therefore perhaps not out of place to mention the work done upon the same symptom by Herz, and to recall that the latter author was able to show that such apical pain was only one symptom of a definite symptom complex, to which he gave the name phrenocardia.

The importance of Herz's work lies in the fact that he was able to definitely show that the apical pain in such patients was not of cardiac origin but arose from a painful spasm of the diaphragm localized in the apical region. Perhaps equally important was the further fact shown by Herz that most of the patients exhibiting this syndrome were women who ceased to be tormented by this pain upon the cessation of their sexual life. Herz's work, having withstood the test of reinvestigation, might be held as vindicating the viewpoint of those who do not attach too grave a significance to pain arising in the apical region.

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Doctor Brown (closing).—The fact that blood pressure does frequently rise in an attack of angina pectoris is not generally considered of special importance, because nearly every writer on the subject admits that true attacks occur in which it does not rise. This has been our experience. Whether it rises or not may be due to the condition of the coronary arteries, since in true blocking it does not rise unless the thrombosis occurs in one of the smaller vessels in which case it may rise.

Admitting with Doctor Kaufman that Lewis is right and that symptoms essentially identical with those associated with true angina may occasionally occur in paroxysmal tachycardia, I can only state that in a large number of such cases, one of whom has had an average of one hundred attacks a year for fourteen years under my care, there has never been such a symptom. On the other hand one of our angina cases operated on later by sympathectomy had developed paroxysmal fibrillation coincident with the onset of angina. In other words the substance of what Doctor Bloomfield states underlies the whole study of heart pain. One must know as clearly as possible what the condition of the heart is, what excites the attacks, as well as what arrests them. Doctor Nuzum's reference to thrombosis is interesting especially in view of his careful and extensive study on that subject. We have had two patients with fusiform aneurysm of the descending aorta whose attacks of angina resemble coronary thrombosis. One was relieved by novocain injections in the paravertebral nerves.

THE LURE OF MEDICAL HISTORY

MARCUS AURELIUS SEVERINUS (1580-1656)

A Contemporary of Harvey, and Author of the First Work on Comparative Anatomy

By Frederick Leet Reichert, M. D.
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LAST year the three hundredth anniversary of William Harvey's *De Motu Cordis* was celebrated. It might be of interest to consider a contemporary of Harvey, Marcus Aurelius Severinus, an Italian who abandoned law to study medicine under illustrious native masters, who taught anatomy and surgery, and who became a celebrated teacher and surgeon of Naples, writing extensively on a variety of subjects, but now being celebrated in the history of medicine as the author of the first comparative anatomy (*Zoötomia Democritae,* 1645). This work, though crude, antedated the writings of Malpighi, Leeuwenhoek, and Swammerdam.

THE SEVENTEENTH CENTURY, THE AGE OF SPECIALIZED ANATOMICAL RESEARCH

The dawn of modern anatomy in the sixteenth century, with the actual public dissections of the human body, and the publication of Vesalius' *Fabrica* in 1543, was followed in the seventeenth century by extensive specialized anatomical research. Quite naturally Severinus made anatomical investigations, but in his eagerness for research he utilized the more accessible bodies of animals. This research led to the publication of the *Zoötomia* in which the woodcuts show the viscera of birds, fishes, and mammals with some comparative phases of their development.

SEVERINUS, THE SURGEON

Both as an anatomist and as a surgeon, Marcus Aurelius Severinus was highly esteemed by the populace, and for a long time he filled the chair of anatomy and later was professor of surgery in the